CALCIFIED CHRONIC SUBDURAL HEMATOMA - CASE REPORT

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The authors present a case of a female patient, 26 years old, who suffered a craniocerebral trauma 15 years ago, by falling from a different level. She manifested an intracranial hypertension syndrome and seizures at the moment of hospital admission. Brain CT scan reveals a large right fronto-parietal calcified subdural mass with left shift of median line. The lesion was isointense on T1 and hyperintense in T2 weighted magnetic resonance imaging (MRI). Calcified or atypical meningioma, bony tumor and subdural abscess were included in differential diagnosis. She underwent surgery and a calcified chronic subdural hematoma was completely excised, with good postoperative recovery. After two months of follow up, seizures were controlled with a minimal dose of medication, control CT scan showing absence of the hematoma and brain re-expansion. In summary, this uncommon calcified chronic subdural hematoma was successfully excised, resulting in a good recovery. From the literature review and the experience in this patient, authors consider surgical treatment for calcified chronic subdural hematoma as necessary and often results in neurological improvement, especially concerning seizures.

Keywords: calcification, chronic subdural hematoma, seizures

INTRODUCTION

Calcified chronic subdural hematoma (CCSH) is relatively common since the first description in 1884 (8). In the elderly, observation is recommended for asymptomatic calcified chronic subdural hematoma without acute or progressive neurological disorders. The incidence of CCSH has been reported to be 0.3% to 2.7% of all chronic subdural hematomas (CSH) (8). There are about 100 cases reported in the literature (10). Although surgical treatment for the CSH is widely accepted, there is still some controversy about whether it should be used.

CASE REPORT

The authors present a case of a female patient, 26 years old, who suffered a cranial-cerebral trauma 15 years ago, by falling from a different level. She manifested an intracranial hypertension syndrome and seizures at the moment of hospital admission, which started in January, 2009 and persisted under

anticonvulsant treatment (carbamazepine 2/day).

Brain CT-scan (native and bone window) reveals a large hypodense elliptical right frontal-parietal calcified subdural mass, with left shift of median line (Fig. 1a, 1b). The lesion was isointense on T1 and hyperintense in T2 weighted magnetic resonance imaging (MRI) and had a volume of 7/6/1,2 cm (Fig. 2a, 2b). Computerized EEG shows a theta-subtheta lesion of 6 c/s, with right temporal localization, which tend to irradiate controlateral.

Calcified or atypical meningioma, bony tumor and subdural abscess were included in differential diagnosis.

After complete investigation, the patient was proposing for surgery, in attempt to total removal of the compressive mass, to establish a histopathological diagnosis and to eliminate the seizures. The surgery was performed under general anesthesia, with the patient in supine position and the head fixed in Mayfield frame, raised at 200 and controlateral rotated at 300.

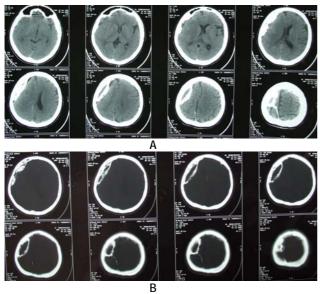
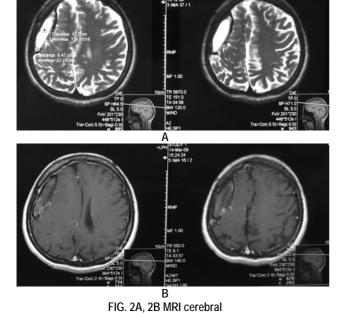
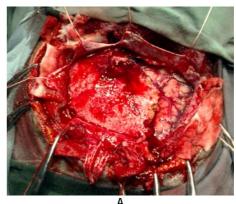


FIG. 1A, 1B Preoperative brain CT-scan (native and bone windows)



A frontal-parietal flap was made and the dura mater was opened in an arcuate manner, with basal pedicle and then was suspended. An elliptical mass of about 7/6 cm was found, with hard consistence and liquid

yellow content (Fig. 3a, 3b, 3c). The calcified hematoma was then totally removed (Fig. 4).





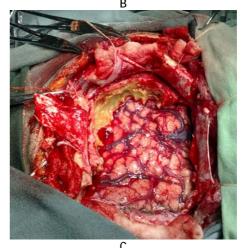


FIG. 3A, 3B, 3C Intraoperative picture reveal calcified hematoma

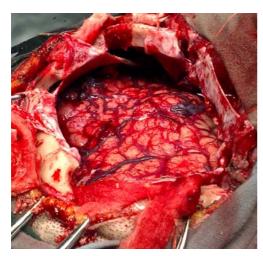


FIG. 4 Intraoperative picture after the calcified hematoma was then removed

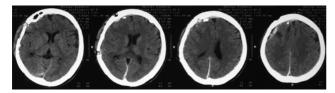


FIG. 5 Postoperative CT-scan 7 days after surgery

Hemostasis was achieved by electrocoagulation and surgicel. Wax was added to bone. The dura was closed with nylon 3.0. An epidural drainage was applied. The bone flap was reinserted. The skin was closed in anatomical layers. The wound was patched. No complications occurred during the procedure, neither after surgery. Postoperative the patient was conscious, cooperative and without motor or sensitive deficits. The drainage was removed 48 h after surgery and the patient was mobilized. Anticonvulsant treatment was maintained.

Control CT scan (Fig. 5) was made 7 days after surgery, showing absence of the hematoma and brain re-expansion. At this time surgical threads was suppressed and the patient was checked out with the following instructions: follow up at 2 months, continue anticonvulsant treatment at least 6 months, avoid alcohol, coffee and sun exposure.

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Histopathological examination reveals chronic subdural hematoma with fibrous transformation and large areas of calcification.

Long term prognosis is excellent.

DISCUSSION

Chronic subdural hematoma is usually one of the complications of minor head trauma. Disorders such as coagulopathy (therapeutic anticoagulant use, sepsis, liver insufficiency), intracranial hypotension (secondary to overdrainage resulting in bridge veins traction in shunted patients), chronic alcoholism, vascular malformations, and primary and metastatic tumors may also play roles in the etiology of chronic subdural hematoma (7). Calcification or ossification is observed at a rate of 0.8% to 10% in patients with chronic subdural hematoma (5). The pathogenesis of calcification in chronic subdural hematomas is not completely clear. However, many authors have suggested that local, metabolic, and vascular factors play a role in the development of calcification and ossification. Afra reported that the circulation in the subdural space with absorption and vascular thrombosis may be responsible for the development of calcification (1).

Most CCSHs are located at the convexity and the extent of the calcification varies widely, sometimes involving the entire hemispheric surface. CCSHs can be diagnosed by CT or MRI (6, 10), and differentiated from the usual CSH by imaging studies and gross pathology. CCSHs have the following characteristics: (1) elliptical shape with the longest diameter in the frontal-temporal direction; biconvex or flat shape on cross section, (2) the content is gelatinous or clay-like, but not liquefied, (3) the inner membrane is thick and has sinusoidal blood vessels, (4) the inner membrane is partially adhered to and evaginated into the cerebral cortex (2-4).

Sometimes the CCSH may be confused with other calcified extra-axial space-occupying lesions, such as calcified:

- epidural hematoma
- · calcified subdural empyema
- meningioma

- · calcified arachnoid
- cyst and
- calcified convexity dura mater with acute epidural hematoma,
- inherent metabolic tendency to calcification, prolonged existence of the hematoma in the subdural space.

The course of the development of calcification in a CCSH is unclear. However, the hematoma may progress gradually from hyalinization to calcification, and finally ossification through irritation of the tissue. After hemorrhage calcification usually takes 6 months to many years to develop (1, 8). It is difficult to understand the mechanisms of the development of a calcified CSH, because it takes a long course. Poor circulation and absorption into the subdural space and vascular thromboses, inherent metabolic tendency to calcification, prolonged existence of the hematoma in the subdural space, stagnant blood due to sufficient arterial supply and inadequate venous return, thick connective tissue membrane, and other local factors are considered to contribute to the development of calcification of CSH (1, 7).

One of the most frequent complications that may be observed after chronic subdural hematoma operations is recurrent hemorrhage. It is thought that insufficient brain expansion following hematoma drainage, developing following prolonged compression in recurrent hemorrhage is the basic factor (9).

CONCLUSION

In summary, this uncommon calcified chronic

subdural hematoma was successfully excised, resulting in a good recovery. From the literature review and the experience in this patient, authors consider surgical treatment for calcified chronic subdural hematoma as necessary and often results in neurological improvement, especially concerning seizures.

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